3. The great majority of patients should not be catheterized. A urinary tract once infected is a constant menace to both the well-being and the life of the patient.

4. New and increasing signs of damage to the cord indicate the necessity for active treatment. X-ray evidence of bone or foreign body pressing on the cord suggests the advisability of operation.

5. Compression of the spinal cord impairs the venous and arterial circulation. This, in turn, retards repair, and may further jeopardize fibers and cells of the cord that are still viable.

6. The Queckenstedt test is our best means of determining whether the cord is compressed, and a positive test is one indication for manipulation or operation.

7. The use of the Queckenstedt test at the time of manipulation or reduction of a dislocation of cervical vertebrae is extremely helpful.

384 Post Street.

DISCUSSION

FREDERICK LEET REICHERT, M.D. (Stanford Hospital, San Francisco).—Doctor Fleming's article on the injuries of the spinal cord and their treatment emphasizes the importance of careful examination and an early diagnosis. The use of the Queckenstedt test, as an aid in determining whether a severe injury to the cord has led to a complete block, and particularly the use of this test after the reduction of dislocated cervical vertebrae is an excellent adjunct in deciding whether operative interference is indicated.

One cannot emphasize too strongly the fact that once paraplegia has developed, following a spinal cord injury, pressure sores will occur within just a few hours.

The use of hot-water bottles to overcome shock seems to do more harm than good in such cases, since the burns that develop become major factors in treatment, and the treatment of shock by other means would have led to less disastrous results.

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EDMUND J. MORRISSEY, M.D. (234 Medical Building, San Francisco).—The plan outlined by Doctor Fleming for the treatment of spinal cord injuries is excellent. It emphasizes the importance of nursing care and the proper treatment of the bladder, and is neither too radical nor too conservative.

There is no subject in the field of neurological surgery in which there is such a wide divergence of opinion. This will continue because we are unable to distinguish, in the first few weeks, between an anatomical and a physiological interruption; and when improvement does follow surgery it is impossible, as a rule, to say with any degree of certainty that such betterment is the direct result of the operative intervention.

There is no doubt that in the great majority of cases the damage takes place immediately as a result of direct injury and, therefore, the results of surgery are disappointing. The conditions which might justify operation are, first, a block as demonstrated by the Queckenstedt sign, which is not relieved by reduction of the dislocation; second, impingement into the canal of fragments of bone as demonstrated by x-ray; and third, increasing neurological findings.

Operation offers more hope when the lesion involves the cauda equina. In one case I found the dura torn and one of the edges with two posterior roots caught in between the fracture of the lamina.

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MARK ALBERT GLASER, M. D. (727 West Seventh Street, Los Angeles).—Doctor Fleming has carefully emphasized the importance of the jugular compression test, and has added a very valuable addition to be used in cervical and cauda equina injuries. These new points that Doctor Fleming brought up are extremely ingenious, and one cannot compliment him sufficiently for such keen observations.

Spinal concussion is quite possible, and may be compared with cerebral concussion. The results of spinal concussion with edema and pin-point hemorrhage would be a complete paraplegia. Personally, I have had the opportunity of seeing several cases wherein the bullet wound grazed the vertebral column, but did not cause a dislocation. A few of the particles of lead were visible along the body of the vertebra where the bullet had touched. The result was a complete paraplegia. The jugular compression response was normal, and there was no blood upon spinal puncture. From this we may assume a cord edema with pin-point hemorrhage.

In view of the dehydrating effects of glucose and other solutions in cases of head injury, we thought it advisable to carry out such therapeutic measures with the hope that some good could be accomplished.

Spinal injury also simulates skull injury in that the damage to the underlying nervous tissue is not always related to the bony destruction. Furthermore, sequelae arising secondary to spinal injuries which are not based upon any organic pathology are extremely incapacitating. The severity of the injury has no relationship to the development of the various types of backache that may occur.

Additional measures must be directed to the treatment of such signs, and efforts made to combat the existing neurosis. Often it is impossible without a *subrosa* investigation to clinically differentiate the malingerer from the psychoneurotic.

MALARIAL TREATMENT OF GENERAL PARESIS*

By F. J. VAN METER, M. D.
Norwalk

DISCUSSION *by Clifford W. Mack, M.D., Livermore; Samuel D. Ingham, M.D., Los Angeles; G. Creswell Burns, M.D., Compton.*

MALARIAL treatment of neurosyphilis has long ago passed the stage of experiment and doubt, and has established itself as a standard remedial measure, as a result of the encouraging reports concerning its use from all over the country. No disease, perhaps, has presented such an utterly hopeless outlook as general paresis until the advent of the arsenicals; and even then, with that advantageous treatment, the results were not all that could be desired. However, admittedly there was a general improvement in a certain group of those who received the drug treatment.

Now, inoculation with malaria has gradually brought an increase in the number of improved patients beyond that seen as a result of treatment with arsenicals, and is, therefore, due to receive such credit as it may. This is borne out by the experiences of many men everywhere, and the literature is replete with improved percentages, all the way from 15 to 35 per cent—something unknown and scarcely hoped for in the years gone by.

CLINICAL MATERIAL STUDIED

In December of 1926, a small group of seven patients in the Norwalk State Hospital were in-

* Read before the Neuropsychiatry Section of the California Medical Association at the sixty-third annual session, Riverside, April 30 to May 3, 1934.

oculated with tertian malaria. The results were not encouraging at that time; on the whole, however, one of this group of men is still living, moderately improved, but not well enough to leave the hospital. The others have passed away at varying lengths of time, but not as a result of the treatment. We found these patients were too far advanced in paresis, and too much impaired physically to be good subjects for the treatment. It may be said that, subsequently, more care was used in selection of subjects, and a year later a more stabilized improvement was noted.

In a group of forty-nine patients treated a year after the inception of the use of malaria, about 12 per cent exhibited an encouraging improvement in that there was a disappearance of mental symptoms, such as delusions, irritableness, combativeness, and the like. The serology, however, exhibited little change, and it was estimated at that time, and statements so made, that the treatment probably was not going to affect the serology. Depressed paretics were believed to respond better to the treatment.

Negro patients were found largely immune, and that is still our viewpoint, following the treatment of as large a group as six hundred. This is comparable to the experiences of others with the negro race. Likewise, Filipinos do not respond to the inoculation many times. While it is possible in some instances to learn that the patient has had a previous attack of malaria, and likely has an immunity, in others there is no history of a previous attack.

In the latter part of 1928, a group of 148 patients had been treated, and on investigating the condition of these patients it was discovered that the Wassermann still remained unchanged largely, after a period even of two years, but the cell count in the spinal fluid was reduced, sometimes to normal, and the globulin was negative. It will be noted here that it was first assumed that there was no change to be expected in the serology, based on our earlier experience, and on the smaller group. General improved condition among this larger group of patients, with apparent remissions, were more frequent, and 15 per cent showed a more durable and lasting improved state. It was felt, as a result of two years' experience, that opinions relative to the efficacy of the treatment should be deferred until six months to a year, or more, after the treatment had been completed.

COMMENT

It is true that, clinically, the patients sometimes improved within a few weeks; but the patient and his relatives, and of course the physician, are particularly interested in the serology, and many times judged the treatment according to what effect it had on the Wassermann. While it naturally is a desirable result to attain lessened positivity, the patient may reach a considerable degree of improvement clinically, and his Wassermann remain unchanged, or at least altered little. We are not treating the Wassermann, we are treating the patient. Some of those patients who after prolonged treatment have exhibited a negative Wassermann on the spinal fluid are steadily deteriorating, while others are improved who remain positive. The fond relatives expected that some miraculous change would come about immediately the treatment had been given.

The public prints wrote up the treatment as a cure for insanity, which it was not, and is not. We should be guarded in giving opinions concerning what malaria will do, and consider the form of neurosyphilis and its degree of advancement before giving a prognosis.

LATER RESULTS

In December of 1929, two hundred and thirty-four patients in all had been treated as described, with the result that we found 25 per cent of them, approximately, markedly improved, or in temporary remissions, and of these, about 14 per cent were able to leave the hospital, but on parole; none were deemed recovered, and there is no information submitted to the institution that they have entirely recovered their former mental health. However, there is no question of their genuinely improved state. The unimproved group of the 234 patients totaled 15 per cent. Now, in this unimproved group little results were shown: the patients had no change in the serology, continued to deteriorate mentally, were deluded, irritable, combative, and showed the general progressive debilitation of paresis. Among the improved there was noted still a positivity of the Wassermann, but a negative globulin, and a cell count normal, or nearly so. Two patients of the 234, on the other hand, showed a slight reduction in the spinal fluid Wassermann, which elicited the hope that later on there might be some alteration.

A group of four hundred patients had been treated by November of 1931, and the markedly improved and stabilized patients in this group amounted to from 22 to 25 per cent. It was found that the manic, or excited and expansive type of paretic, responded much better to the treatment than did depressed patients, which is just contrary to our view of that matter four years before.

At this time we are considering a group of six hundred patients, who have been treated since December of 1926. Without going back into figures and statistics too much, this whole group showed approximately 34.3 per cent, or 206 patients in the improved status; 40 per cent, or 240, after everything is considered with reference to condition, were unimproved; 154, or 25.6 per cent, died within from two weeks to two years after treatment. Now, in speaking of patients who are improved, the writer is indicating that these patients had a disappearance, gradually, of active mental symptoms, deluded states, hallucinatory experiences, marked irritableness and combativeness, with reduced to sometimes normal cell count, with negative globulin, and a slight lessening in the positivity of the Wassermann. Since 1926 the improvement appears to have gradually advanced from a point of about 12 per cent on up to the present figure, and these averages, as compared with others using malaria, appear fair.

NOTES ON THERAPY AND PROCEDURE

It has been observed that women patients terminate the malarial attacks spontaneously many times, and much more frequently than men. In fact, it appears that the spontaneous termination of the women is more the rule than the exception. With the men in our group, it is unusual to have such a termination.

The same strain of malaria has been used at Norwalk since 1926, and is the tertian variety, the paroxysms of chilling and febrile rise occurring every day. Quinin has terminated malarial attacks without fail, though occasionally there may be a recurrence which another course of quinin stops. In some individuals a follow-up course of neoarsphenamin or sulpharsphenamin has been given instead of quinin, which seems to work satisfactorily in terminating the malaria, and in providing the necessary tonic and alterative effect. In general, the follow-up treatment is supportive and tonic. Tryparsamid, neoarsphenamin, or sulpharsphenamin is used.

The patients who show arteriosclerotic changes, valvular heart disease, myocardial disease, nephritis, pulmonary disease, or marked general debility from any cause, are not good subjects. The arteriosclerotic group particularly do not go through the treatment well.

These patients are allowed to have a course of about fifteen chills and febrile attacks, and throughout their clinical condition are closely watched. The patient must be treated, and not just the disease essentially. The appearance of cachexia, general weakness, marked jaundice, are some of the indications to interrupt the malaria. We have to keep in mind that we are treating an individual already depleted by a grave disease, paresis, and that we have inflicted him with another disease, even though the object is remedial. In the well-selected subjects not weakened physically, or possessing some of the ailments pointed out heretofore, the treatment does not greatly deplete the patient; and between the chills and fever elevations, the patient appears to feel generally comfortable. If the fever mounts to a high point, the ice cap and plenty of fluids will render comfort, as well as the application of warmth during the chills. It is our experience that the temperature will range from 102 to 106 degrees. With a higher temperature, there may be some muttering delirium, restlessness or excitement, subsiding after the temperature goes down. Some patients had a very mild chill and mild febrile rise.

TECHNIQUE OF THE TREATMENT

Briefly, as to the technique of the treatment, one and one-half to two cubic centimeters of blood is drawn from the vein of the donor during his attack of tertian malaria, the blood being mixed with about one cubic centimeter of sodium citrate solution one per cent, and this mixture is injected into the vein of the patient. The subcutaneous method has not been used. We have had no instances in which death has followed shortly after the inoculation. The incubation period has varied from four days to ten, and even as long as twenty-one days, the average being approximately a week. The patients are put on a four-hourly clinical chart the fourth and fifth day after inoculation, and are kept closely under observation. The hour at which the attack comes is very carefully noted, since this may vary from day to day. The attack of fever comes daily as a rule, though there may be a variation as to its height on alternate days. The patient during the attack of shivering is made as comfortable as possible in blankets with local heat applied if necessary. During the sweating stage, the clothing may need to be changed. Liquids are freely given. There is a tendency toward constipation, and this must be corrected. Albuminuria may develop. If it is severe, the attack should be stopped. The blood for inoculation may be taken from the patient at any time as long as he has the malaria, and not necessarily during the febrile attack.

Blood examined from a patient during a chill shows a mature form of the malarial parasite, together with some younger forms. Parasites at different stages of development are seen. During the chill there appear two predominating types of the mature form, and the younger forms, which possibly accounts for the chills occurring every twenty-four hours. Examination of the blood in the same patient some twenty hours later following the chill, while it still shows many of the parasites in various stages of development, exhibits the older forms predominating with few of the young forms being seen. It is well to examine the blood from time to time, since it serves as a reliable guide along with the clinical signs of the progress of the disease.

IN CONCLUSION

With reference to just what factor is bringing about the betterment in these patients, it has been assumed and believed, with just cause, that the elevation of temperature accompanying the malarial attack was responsible for the benefits derived. Some hold to the view of a biochemical reaction coming about as a result of the malarial infection. Some of our patients who have had just mild chills and not a particularly high fever during their experience with malaria have enjoyed as substantial improvement as those with hyperpyrexia. Some patients who had extreme high temperatures have not seen as good improvement as those with milder fever. Speaking generally, it appears to us at this time—viewing this whole group of six hundred—that the patients who experienced the higher temperatures have had the better results. The exact nature of the process which brings about the improvements and remissions cannot definitely be assigned. The malarial attack may inhibit spirochetal activity in some manner not clear, aside from the thermogenic angle.*

SUMMARY

1. Spirochetes found by the Jahnel method in brains of six out of thirty-nine malaria-treated paretics.

* Spirochetal findings in brains of paretics treated with malaria, by Nicholas Kopeloff and Nathan Blackman, Department of Bacteriology, Psychiatric Institute and Hospital, New York City; American Journal of Psychiatry, July, 1933, pages 21 to 30.

2. Among six positives, degenerate forms of spirochetes encountered in more instances than normal forms. Three of six patients come to autopsy in six weeks after inoculation.

3. Spirochetes found by Jahnel and Dieterle methods in eight of ten paretics not treated with malaria.

4. Artificially induced malaria is likely to destroy spirochetes in brains of paretics; or, failing to destroy spirochetes completely, this therapy alters morphology of spirochete to such a degree as to render it degenerate in appearance.

Norwalk State Hospital.

DISCUSSION

CLIFFORD W. MACK, M. D. (Livermore Sanitarium, Livermore).—The subject is covered in the author's paper in a very comprehensive manner. The large group of four hundred patients treated makes it possible to draw valuable statistical conclusions. It is stated that from 22 to 25 per cent of the patients are improved and stabilized. This accomplishment in the treatment of paresis, which has for years been considered well-nigh hopeless, is very noteworthy.

Another point brought out in the paper which should be emphasized is that, although there was not much improvement in the serology, nevertheless clinical improvement is the valuable thing and warrants the writer in stating that they are treating the patient and not the Wassermann. We have positive evidence that the malaria is efficacious in paresis by the autopsy findings. The article by C. Geary of the Central Pathological Laboratory of the London County Mental Hospitals cites several cases in which histological examination showed none of the characteristic findings in the brain tissue of paresis after the malarial treatment. These cases were positively diagnosed as paresis.

We should endeavor to obtain the consent of patients and relatives to administer the malarial treatment in the preparetic stage of neurosyphilis before degenerative processes have begun. This can best be accomplished by periodical spinal fluid examination in known cases of lues, so that the asymptomatic stage may be recognized and treatment administered accordingly.

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SAMUEL D. INGHAM, M. D. (727 West Seventh Street, Los Angeles).—The results obtained by the treatment of general paresis in a State hospital, where the patients as a rule are in a relatively advanced stage of the disease, are not a true indication of the value of the treatment. It is obvious that the best results from any treatment can be obtained in an early stage of the condition treated, but it is seldom that patients are committed to the State hospital in an early stage of general paresis. It is very significant, therefore, that more than 33 per cent of a series of six hundred patients in the Norwalk State Hospital are definitely improved following the malarial treatment. The fact that these results correspond to the results from many hospitals and clinics has established the method as a distinct advance in the therapeutics of neurosyphilis. Although the author did not mention the after-treatment, it is well to remember that, after a patient has been treated by malarial inoculation or by other forms of hyperthermia, this should be followed by a prolonged series of treatments by bismuth and tryparsamid, checked by periodic examinations of blood and spinal fluid.

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G. CRESWELL BURNS, M. D. (Compton Sanitarium, Compton).—In the past five years at Compton Sanitarium, we have treated twenty-six paretic patients by malarial inoculation. Twenty-four of these were patients who showed clear-cut symptoms of general paresis, while two were classified as belonging to the group of vascular central nervous system syphilis. Only three of the group were women. With such a small group statistics relative to percentages of recoveries, improved and unimproved states, can have little value. Some of these patients came to the sanitarium in the last stages of the disease. Some had had little or no antiluetic therapy prior to admission. Most of these patients were admitted soon after they had become unmanageable at home. In several instances symptoms of insidious parenchymatous involvement were present many months, occasionally two or three years, before a physician had been consulted. Five of our inoculated patients died. All but one of these were poor risks to start with, and the probability of a fatal outcome was explained to the relatives. The deaths were in the male group. No autopsies were obtained. One patient, forty-six, died from a cerebral hemorrhage the day following inoculation with malaria; so we do not feel that malarial inoculation was responsible for this death. In the other cases death occurred from eight to twenty-one days after inoculation. These four developed a terminal acute nephritis. One of these patients was thirty-eight, the remaining three over fifty, and arteriosclerotic.

Twenty of our inoculated patients were considered to be improved following the treatment with malaria. Only one was definitely unimproved. Of the improved group, three patients are known to be working. One of these was inoculated in 1929, one in 1933, and the other, a young man of twenty-five whose infection was congenital, was inoculated in February, 1934. He had received considerable antiluetic treatment periodically for several years. The onset of acute mental symptoms was in December, 1933. The young man returned to work this summer. One patient of the improved group became so clear mentally that a confusion over his identity resulted. Following malarial treatment he resented being called by the name under which he was registered. He remembered having a brother on the eastern coast of Canada and investigation resulted in our learning that this patient had been missing from his family for twenty-five years.

The diagnosis of general paresis had already been established in twenty-two of the twenty-six patients before admission to the sanitarium. Two were admitted because of alcoholism, one with a long-standing diagnosis of Parkinsonian syndrome, and the fourth, not referred by a physician, came in undiagnosed. Tryparsamid has been used routinely as follow-up therapy.

Bruetsch (The Histopathology of Therapeutic (Tertian) Malaria, in *The American Journal of Psychiatry*, July, 1932) has shown that therapeutic malaria, in addition to producing a stimulation in general of the reticulo-endothelial apparatus (system of histiocytes) leading to a new formation of macrophagic tissue in various organs, also produces a macrophagic response in the nervous system, greatest in the leptomeninges. To quote Bruetsch: "In the adventitial sheaths of the vessels of the brain cortex, the mesodermal phagocytes are only slightly stimulated. About middle-sized and large cortical vessels a small increase in the number of macrophages has been found. In the perivascular spaces of the large vessels in the white matter, and in the striatum, and in the pons, stimulated histiocytes are more numerous. The small mesodermal elements along the capillaries of the brain cortex have not been activated."

Since encouraging clinical results have been reported in paresis by the use of diathermy and the employment of high temperature baths, whether or not a similar macrophagic response is produced in these patients by fever alone is something for a work such as Bruetsch's to determine. Freeman (*Journal of the American Medical Association*, June 3, 1933), concluded, from necropsy material in six of his cases in his series treated by diathermy, that there was persistent inflammation in every one of the brains examined. But the amount of macrophagic response was not stressed by Freeman in this particular article. It would be interesting to treat a series of paretics with malaria and terminate the malaria after only two or three chills. If definite clinical improvement re-

sulted, it could reasonably be assumed that the macrophagic response was an important factor in the mechanism effecting these remissions. Certainly we do see patients obtain remissions following a poor malaria "take." One of the patients in our series was such an example.

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DOCTOR VAN METER (Closing).—The writer appreciates the discussion by Doctors Mack, Ingham and Burns, with reference to the additional points brought out by them. There can no longer be any question about the efficacy of malarial therapy in the light of all these experiences.

The paper was intended to deal only with malarial therapy and, for the sake of brevity, the after-treatment with the arsenicals and bismuth was not gone into. I may say that the patients afflicted with general paresis in the State hospitals, at least so far as Norwalk is concerned, are not all deteriorated, and some of them are not so far advanced, and I feel that our findings and work are very much of value in view of this. No doubt, the best results can be obtained by treatment in the early stages; however, what appears to be deterioration is not always such, or we would not have had such encouraging results.

HERPES ZOSTER—SOME OF ITS CLINICAL ASPECTS*

By MELVIN R. SOMERS, M. D.
AND
PEARL S. POUPPIRT, M. D.
San Francisco

DISCUSSION *by Samuel D. Ingham, M. D., Los Angeles; Merlin T.-R. Maynard, M. D., San Jose; Hiram E. Miller, San Francisco.*

THE multiplicity of therapeutic measures recommended for herpes zoster points to a lack of basic knowledge for rational therapy of this condition. Rational therapy requires an understanding of pathology and more especially pathological physiology. Therefore, as a basis for a discussion of some clinical results we have obtained in the treatment of this condition by posterior pituitary extract, we shall briefly call attention to some of the pathological and physiological aspects of herpes zoster.

PATHOLOGY AND PHYSIOLOGY

In 1863, Bärensprung [1] first demonstrated that herpes zoster is accompanied by changes in the spinal ganglia. Since that time a great many observers have studied the pathological anatomy of herpes and have established that there are two types of the condition, known as primary and secondary. So-called *primary* herpes zoster is apparently an acute infectious disease which is accompanied by fever and leukocytosis, and has a self-limited course, usually followed by immunity. In this condition there is an acute inflammation [2] affecting mainly the dorsal ganglion of the spinal segment involved. Pathological studies of herpes zoster that occasionally occurs in the course of a great variety of other diseases, demonstrate that there is also a *secondary* type. These studies show that in the secondary type there is a lesion at some point in the reflex arc which extends from the spinal cord through the posterior roots and dorsal ganglia to the peripheral nerves and skin, or the lesion may be at any point in the viscero-sympathetic-spino-radicular arc.[3] Various chemical agents giving rise to herpetiform lesions, such as arsenic and carbon monoxid, have the common property of increasing the permeability of the capillaries.[4] The vascular changes have been so prominent that Peiffer and others have thought that the distribution of the skin lesions follows that of the blood vessels.

If one considers the many sites of the pathological changes found in herpes zoster, it is evident that any theory of the origin of the pain and skin lesions must be consistent with disease at any point along the reflex arc, and even in the blood vessels of the skin itself. The observation by Stricker [5] that vasodilatation occurs in the distribution of the posterior spinal root, when its peripheral cut end is stimulated, has been amply confirmed by Bayliss,[6] Langley,[7,8] and others. It is not within the province of this paper to go into the exact mechanism by which the capillary and arteriolar dilatation is produced. Vasodilator fibers may be stimulated acting directly upon the peripheral blood vessels, or the action may come about from antidromic impulses. According to Bayliss, it is the skin rather than the other tissues of the neural segment which is chiefly affected in the vasodilatation originating in antidromic impulses.[6] Lewis [9] has brought forth experimental evidence to show that the nerves do not act directly upon the skin vessels, but that there is a chemical substance resembling histamin in its action, liberated peripherally, and that this is responsible for the cutaneous capillary vasodilatation. The existence of a vasodilator substance is not accepted by everyone. It is possible, as has been suggested by Lewis [9] and others, that the arteriolar dilatation accompanying the capillary dilatation may come about as the result of an axon reflex. Anatomical connections have been traced between the nerve-endings and the adjacent blood vessels. Clinical and some experimental observations show that prolonged stimulation of sensory nerve fibers is accompanied by vasodilatation, and produces swelling, chemosis, and vesicle formation in the innervated area. That the vasodilatation, swelling and vesicle formation is accompanied by local irritation of sensory nerve-endings is a matter of common clinical observation. This type of pain may be relieved, as Bleuler [10] recommends, by the use of cocain ointment upon the lesions, demonstrating that it is due to peripheral rather than more central irritation. It is plausible, therefore, that once vasodilatation and chemosis have occurred, a local axon reflex takes place and adjacent blood vessels open up. Thus a vicious circle would be established, limited only by the intensity of the vasodilator stimulus either direct, antidromic, or reflex and the amount of vasodilator substance liberated, if one exists. The irritation of sensory endings by the local reactions preceding and accompanying the herpetic lesions is probably responsible for the type of pain with which

* From the Division of Neuropsychiatry of Stanford Medical School.

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